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Effects of Rust on Plant Development in Relation to the Translocation of Inorganic and Organic Solutes

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I. Introduction

The interaction between a cereal and a rust fungus leads to many diverse changes in the growth and development of the host. One of the most profound of these changes is in the distribution patterns of inorganic and organic solutes. The significance of this change arises from the proposition that (1) the distribution of solutes constitutes "a key factor in productivity" (Loomis *et al.*, 1976), and (2) cereal rusts create major imbalances in these distribution patterns. The evidence emerging for this proposition is largely indirect but compelling. Actually, observations bearing on this view have been accumulating for many

years. Cornu (1881), for example, was commenting on this when he spoke of the "activité vitale" of rust infections, and even earlier reports can be found in the writing of medieval herbalists; stunting of cereals, presumably due to rusts, is even mentioned in the Bible.

Because rust fungi are biotrophic, they have had by necessity to evolve ways to superimpose themselves upon the host without killing it—at least in the beginning of the infection cycle. Second, because they cannot adequately develop throughout their life cycle solely at the substrate levels found in parasitized cells, they also have had to evolve mechanisms for obtaining nutrients at some distance away from these cells. How this comes about is the topic of this chapter.

II. Distribution of Solutes during Plant Development

To appreciate how rust diseases affect solute distribution patterns in cereals, it is first necessary to understand the patterns of metabolite movement in healthy plants, as well as the underlying mechanisms responsible for them (Lüttge and Pitman, 1976; Stocking and Heber, 1976; Zimmerman and Milburn, 1975). These patterns vary from one metabolite to another and depend on many factors [e.g., developmental stage of the plant, external stresses]. Furthermore, they can change within minutes if the external conditions change (Fondy and Geiger, 1980; Geiger, 1976; Wyse and Saftner, 1982).

Some inorganic solutes are extremely mobile, as exemplified by phosphorus and potassium, taking part in reactions in one cell, then likely as not being translocated to another cell—either nearby or relatively distant—where the process is repeated. Others, calcium for example, are the antithesis of this. Once it enters the cereal leaf, it appears to remain essentially immobilized until the leaf dies. However, a considerable amount of the calcium that remains in the stem may ultimately be transported to the developing grain (Martin, 1982).

Organic compounds also are in a continual state of flux, their rates of turnover [relative rates of synthesis and degradation] depending on the compound in question. However, essentially all compounds that have been carefully studied have been found to turn over, even those secondary products that once were thought to serve only as metabolic end products for disposal of toxic substances. Thus an individual carbon or nitrogen atom may sequentially be a constituent of a large array of compounds within a single cell before passing on to another cell. It is only when senescence of the leaves and maturation in the grain itself

occurs that metabolic turnover slows and essentially stops in these organs.

Translocation patterns within the plant are governed by "sources," that is, regions that export solutes and water, and "sinks," regions that import solutes and water for metabolic utilization or storage (Loomis *et al.*, 1976; Sutcliffe, 1976; Wareing and Patrick, 1975). Meristems in vegetative and reproductive organs are the principal sinks, but storage pools and regions of high respiration are also important sinks. The activity of these sinks is determined by many factors, as cited previously; some are intrinsic to the sink itself, whereas others are determined by the sources and/or environmental conditions. In any event, sinks play a large role in determining the plant's priorities for the distribution of solutes and water.

During germination, the seed's endosperm initially acts as the major source for inorganic solutes, but soon the root system takes over this function. Although the roots continue to obtain and translocate inorganic solutes throughout the cereal plant's life, it is important to note here that as the plant body develops, an increasing proportion of these solutes entering the shoot meristem, and ultimately the grain, are translocated from older leaves and the stem rather than directly from the roots [Durbin, 1967]. In wheat, for instance, Martin [1982] found that 75% of the nitrogen, 86% of the phosphorus, 22% of the potassium, and 37% of the magnesium in the vegetative portion of the plant were translocated into the grain. In oats, more than 90% of the phosphorus and nitrogen of the grain has been accumulated by the plant before it reaches 25% of its maximum dry weight (Williams, 1955). Likewise, in corn 60% of the kernel nitrogen comes directly from the leaves (Hay *et al.*, 1953).

The leaf blade, together with the sheath that covers most of the stem in cereals and, to a greater or lesser extent, the glumes, awns, and stem, produce most of the organic solutes (i.e., photosynthates and from them the primary and secondary metabolites) (Durbin, 1967). These are either metabolized *in situ* or exported elsewhere, particularly to the developing root and shoot apices. The lower leaves tend to translocate most of their organic solutes basipetally, whereas upper leaves translocate them acropetally.

Nitrogen in the form of nitrate, certain amino acids, and asparagine and glutamine is first supplied from the root to mature leaves via the xylem. Some of the nitrogen is then loaded into the phloem and retranslocated, chiefly as amides and amino acids, to the developing shoot and root. The nitrogen-containing compounds entering the developing shoot are thus thought to have come mainly from mature

leaves rather than directly from the root because, having a relatively small surface area, the developing shoot's requirements cannot be met by transpirationally derived nitrogen. A large proportion of the nitrogen is continually being cycled through the plant. In wheat, Simpson *et al.* (1982) envision this process as constituting a dynamic nitrogen reserve that can increase or decrease depending on the prevailing source-sink relationships.

Solutes entering the leaf are partitioned in various ways, depending on the kind of solute and the stage of plant development. Some are transferred readily from the xylem to the phloem for retranslocation out of the leaf (Pate, 1975, 1980). Others are retained in the leaf either in storage pools or actively utilized in anabolic processes. The path of solute movement within the leaf is not definitely known. Two pathways appear to be probable candidates: an apoplastic one within the aqueous continuum of the leaf's free space (i.e., cell wall) and a symplastic one (i.e., metabolic space) through the plasmodesmata between the mesophyll cells and the sieve elements (Läuchli, 1976; Spanswick, 1976). The prevailing view currently hypothesizes a mixed pathway in which solutes are first unloaded from the symplast (phloem or vein tissues associated with the phloem) into the apoplast. They then move in the apoplast to the mesophyll cells, and finally enter the symplast of the mesophyll cell cytosol. The phloem is loaded by solute movement along the same pathway in the opposite direction (Geiger, 1976; Madore and Webb, 1981). As an example of this, Kuo *et al.* (1974) have suggested that in wheat during phloem loading there may be a movement of sucrose into the apoplast from within the mestome sheath that surrounds all the longitudinal veins. Phloem loading from the apoplast has also been demonstrated in corn (Cronshaw, 1981), and there is evidence that such loading is an active process (Heyser, 1980).

As the plant grows, each leaf goes through a developmental cycle in which, when juvenile, it behaves as a sink; then, as growth slows and maturity approaches, it increasingly serves as a source. A period of maturity follows during which the leaf acts as a net exporting organ. Eventually, it declines in metabolic activity (i.e., becomes senescent), and then dies. During senescence, reserves and proteins are hydrolyzed and, along with mineral nutrients, retranslocated to developing tissues so that, when the leaf dies, much of its nonstructural components have already been transported elsewhere.

Changes in inorganic solute levels in cells can also regulate organic solute movement to some extent. A mild potassium deficiency, for instance, will cause an accumulation of photosynthates in leaves and, conversely, in excess it can stimulate translocation. These effects ap-

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pear to be mediated via an interaction of potassium with a membrane-bound ATPase, which presumably is involved in phloem loading (Giacinta, 1979). To complicate the matter, carbohydrate movement, in turn, appears largely to control phosphorus movement (Marshall and Wardlaw, 1973).

Beginning early and continuing at an increasing rate throughout the plant's vegetative phase, inorganic and organic metabolites begin to accumulate or be produced at levels above those needed for metabolism. In cereals, most of these metabolites are sequestered in stem tissues in the form of secondary metabolites or in storage pools. Subcellular sites for such temporary storage are found in the chloroplast, cytosol, and vacuole. There they remain until after anthesis, when they are mobilized and retranslocated to the developing grain.

During plant growth, the solute distribution patterns, governed by all these source-sink relationships, become more complex as additional sources and sinks are formed. The patterns and the role of the xylem and phloem in translocation change in a series of timely transitions. They undergo their most radical alterations after anthesis, when the developing grain begins to supersede all other sinks. At this point, further vegetative growth almost ceases, the plant increasingly directs its metabolic resources toward grain development, and the grain ultimately becomes, for all practical purposes, the sole sink.

III. Effects of Rust on Solute Distribution

The impact of a rust on this multivariate, dynamic, and adaptive system is manifold and profoundly affects the plant's subsequent growth and development. Such factors as the stage of plant development at the time of infection, the cultivar, the presence or absence of stresses (e.g., other diseases or insect infestations), infection type, disease intensity, location of the infection(s) on the plant, and environmental conditions can modify the magnitude of the effect. The rust has a skewed effect on the host, such that (1) low disease levels have disproportionately large effects, and (2) imbalances created during early growth can have a particularly large effect, as has been shown with powdery mildew (Carver and Griffiths, 1981).

Because plant processes are intimately related, alterations in solute distribution patterns induced by localized infections of rust fungi will have physiological and metabolic repercussions in other organs of the plant and at later times in its development. As an instance of this,

Bushnell and Rowell (1968) found that in severely rusted wheat the major reason for shoot desiccation and death was a drastic decline in the root's capacity to provide water. This decline apparently had originally come about because of a decrease in organic solute transfer to the root from the rusted shoot. This example illustrates one of the general effects that rusts have on their hosts: The developmental cycle of the host or one or more of its organs is foreshortened. For this reason, when infection occurs during especially critical periods such as anthesis or grain filling, productivity can be drastically curtailed because the maturation period is reduced.

Another instance of this linkage among organs in the diseased plant is exemplified by the effect of rust on the photosynthetic rate of healthy leaves. Livne (1964) found that the rate of photosynthesis of a trifoliolate leaf on a bean plant with infected primary leaves was higher than that of a corresponding leaf on a healthy plant. A considerable amount of evidence has accumulated indicating that the removal of photosynthates from the healthy leaf under the influence of the rust is responsible for this increase (Durbin, 1967; King *et al.*, 1967; Wyse and Saftner, 1982). Hartt (1963), working with sugarcane, first showed that a variety of conditions that lead to sucrose depletion increase photosynthesis. Conversely, when sucrose accumulates, the rate of photosynthesis decreases. Her results also help to explain why photosynthesis declines in infected tissues (Doodson *et al.*, 1965), for here, there is an accumulation of photosynthates.

It was evident quite early that rust infections must have a significant effect on the translocation patterns of the host. Shaw and Samborski in 1956 stated, "The relative rates of transport into and utilization of metabolites within the infection zone may well be an important factor in determining both the degree of development of the parasite and the reaction of the host." This view came about because of observations on infected tissues that showed that (1) they had elevated respiration rates and depressed photosynthetic rates (Shaw, 1963), (2) they increased in fresh and dry weights (Yarwood and Childs, 1938; Bushnell, Chapter 15, this volume), (3) they remained alive longer than surrounding tissues, (4) the host cells (in certain rusts) around the pustule sometimes began to divide (Yarwood and Cohen, 1951), and (5) the growth rate of the rest of the plant decreased (Durbin, 1967).

Later studies using radionuclides (e.g., ^3H , ^{14}C , ^{32}P , and ^{35}S) conclusively showed that both inorganic and organic substances accumulated at the site of infection, an area that Shaw (1963) has called the rust's "field of dominance." At first, this accumulation of metabolites was thought to occur predominantly in the host cells at the site. This

view was buttressed by the then prevalent hypothesis of Allen (1953) that host respiration might be accelerated by an uncoupling agent elaborated by the pathogen. However, additional research showed that this was not entirely the case and that much of the observed increases in respiration rate and weight were due to the pathogen [Bushnell, Chapter 15, this volume; Daly, 1967; Shaw, 1963]. Closer inspection also has shown that as the time after administration of a radionuclide increases, a larger amount of the label is incorporated into the fungal mycelium and spores [Durbin, 1967; Mendgen, 1977; von Sydow and Durbin, 1962].

By administering the radiolabeled substances to different parts of locally infected plants (Doodson *et al.*, 1965; Durbin, 1967; Holligan *et al.*, 1974), it was shown that accumulation was due to two factors: an increase in the rate of solute movement toward the infection site and a decrease in their movement away from the site. Using bean rust as a model system, Livne and Daly (1966) found that rusted primary leaves imported 40-fold more photosynthate from the trifoliolate leaf than did corresponding healthy primary leaves, and Zaki and Durbin (1965) found that photosynthate movement to the stem apex from infected primary leaves is reduced fivefold, and to the root eightfold. [Livne and Daly (1966) found a 40-fold decrease in the latter case.] Studying yellow rust of wheat (*Puccinia striiformis*), Doodson *et al.* (1965) found that ^{14}C translocation over a 3-hr period was reduced as much as 99% from an infected leaf. Thus, much of what appears to be taking place can be explained if one simply assumes that the fungus creates an extremely efficient sink.

The host cells immediately around the infection site also play a role in enhancing the accumulation process. As indicated earlier, these cells begin to photosynthesize less but respire more as the infection develops (Owera *et al.*, 1981; Bushnell, Chapter 15, this volume); thus they become more dependent on surrounding cells for their nourishment. Their starch (Schipper and Mirocha, 1969) and other reserves also are depleted [Bushnell, Chapter 15, this volume], processes biochemically controlled from within the cell (Huber, 1981). An important attribute of cells in this region is that they remain in a juvenile state much longer than would normally be the case. This tends to intensify the sink effect, and, especially in older leaves, minimizes these cells' capacity to act as a source (Durbin, 1967). The net result is that the vegetative and/or reproductive meristems are starved [Doodson *et al.*, 1965; Siddiqui and Manners, 1971]. Just how great an effect this "starvation" has is undetermined, but certainly it is of major consideration, especially during grain development.

These and other kinds of studies clearly show that accumulation at the infection site is at the expense of the remainder of the plant (Doodson *et al.*, 1965; Holligan *et al.*, 1974; Siddiqui and Manners, 1971). Although not designed to measure flux rates of ingress and egress, they did describe overall changes in the translocatory processes. However, it is not clear if these changes involve an increase in the mobilization of reserves followed by their export, or whether there is simply a quantitative change in the solute distribution patterns to the various sinks already present. Also a factor to be considered is the influence rust has on both the photosynthetic and dark CO₂-fixation rates (Rick and Mirocha, 1968) in the various parts of the plant. Quantitative analyses need to be done on these systems using, for example, such approaches as plant growth modeling (Causton and Venus, 1982). This kind of approach has already proved useful in barley for studies on the relationship between brown rust (Owera *et al.*, 1981) or powdery mildew, and yield.

A significant portion of the accumulated nutrients is probably required to support the growth and reproduction of the pathogen, particularly in susceptible combinations where there is a large mass of the pathogen that, during sporulation, rapidly depletes the soluble nutrient pools in the mycelium (von Sydow, 1966; von Sydow and Durbin, 1962). However, we do not know what proportion of these nutrients are metabolically utilized by the pathogen, or what the exact compounds are that traverse the interface between the fungal haustorium and the host plasmalemma (Bushnell, Chapter 15, this volume). Are all these individual chemical species actually required or do some simply accumulate because of mass flow toward the infection site? These considerations are of some moment because, if compounds are non-specifically directed to the infection site and then transferred to the pathogen, then fungitoxic substances could also accumulate within the fungus to concentrations significantly higher than would be found in the surrounding tissues. In essence, the fungus' requirement for nutrients could be turned against it! Such a strategy is the basis for the application of Ni-containing compounds on cereals by low-volume spraying (Peterson *et al.*, 1958).

Although it is thought that the majority of solutes pass into the vegetative hyphae of the pathogen across the haustorial-plasmalemmal interface, there are no detailed quantitative studies showing what proportion of the solutes pass through this interface. On the basis of results with powdery mildews (Bracker and Littlefield, 1973; Heath, 1972, 1976), one might presume that this is the paramount, if not only, portal of entry. Possibly, however, in the rust fungi such exchange may

not be restricted to the haustorial region, but may also encompass a portion, or all, of the intercellular mycelial complex. In support of this, Ehrlich and Ehrlich (1970), using ^{14}C -labeled urediospores, concluded that there is an outward movement of ^{14}C from the pathogen, and that it probably occurs along the intercellular mycelia. Assuming that this movement of solutes could be bidirectional, the host, via the apoplastic space, might be able to provide a significant amount of nutrients to the pathogen. Certainly the water film lining the intercellular spaces and saturating the cell wall (i.e., the apoplast) is replete with inorganic and organic nutrients. In tobacco, for example, the intercellular fluid of leaf tissue contains, per square centimeter of surface area, about 6 μg of carbohydrate, 20 nmol of assorted amino acids and ammonia, and 50 nmol of inorganic solutes (R. D. Durbin, unpublished data). To put this in some perspective, these values would roughly correspond to a very dilute microbial culture medium.

Another factor that helps a rust pustule to become a significant sink is the movement of water. Initially after infection, there appears to be a transient decrease in water loss because of a decrease in stomatal aperture (as measured by an increase in the diffusive resistance of leaves). However, once the epidermis is ruptured by the sporulating fungus, an abrupt and dramatic increase in water loss may occur (Duniway, 1976; Suksayretrup *et al.*, 1982). In contrast to other changes in moisture flux, the plant has very little control over this loss. Such a rapid and localized loss results in an increase in water movement toward the pustule-containing area, an effect that could lead to the accumulation of even more solutes by mass flow. It has been suggested that one way to minimize damage of this type might be to breed for high stomatal resistance (Suksayretrup *et al.*, 1982).

IV. Factors Responsible for Pathogen-Induced Imbalances

One of the major unanswered questions concerns which mechanism(s) are involved in solute accumulation by rust fungi. When considering this question, we need to realize that the process is a continuum involving the directed mobilization and translocation of solutes, as well as their movement across the host-pathogen interface, and their subsequent movement and metabolism within the parasite. Taking this view, it seems probable that a number of mechanisms

acting in parallel as well as in concert contribute to solute accumulation. Unfortunately, detailed information about how rust fungi are able to cause accumulation is necessarily incomplete, because the mechanisms that operate even in healthy plants are not clearly understood. In addition, these fungi, because of their essentially obligate nature (i.e., they adapt poorly to artificial media and lack normal haustoria *in vitro*), are difficult experimental subjects. Furthermore, it is not clear to what extent analogies can be made among the different pathogens causing biotrophic diseases. For instance, although haustoria of the Uredinales share several features with those of the Erysiphales (e.g., structure of the haustorial complex) (Bushnell and Gay, 1978), there are many differences that may alter or invalidate comparisons (e.g., host tissues in which haustoria are formed, kinds and quantity of fungal polyols, and the presence of intercellular hyphae). The evidence presented by Harder and Chong (Chapter 14, this volume) indicates that the haustoria of cereal rust fungi have associated with them a complex of microtubule-like structures extending into the host's cytoplasm. Such structures have not as yet been seen in powdery mildews. Also, Spencer-Phillips and Gay (1981) found differences between the two groups with respect to ATPase activity of the haustorial plasma membranes. Thus rusts may fundamentally differ from the powdery mildews in how their haustoria obtain nutrients from the host.

One may be able to gain some insight into what factors are important in redirecting translocation patterns by considering work done in related areas. Accordingly, listed here are some potentially important factors that might be involved in rust-induced imbalances in solute distribution in cereal plants. As yet, there are very few direct experimental findings to support any one of them. However, this is mainly because appropriate experimental systems have not yet been developed rather than because of any accumulation of negative scientific results.

The ability of rust fungi to delay the senescence of the tissues around the infection site, which otherwise would act as a source for the developing meristems and grain, appears to be one of the major factors responsible for creating imbalances in solute translocation patterns. It is clear that senescence is regulated by a number of interactive processes operating either to promote or retard the process (Thimann, 1979; Thomas and Stoddart, 1980). Accordingly, when rust is superimposed upon this framework, it seems likely that it could affect senescence at one or several points by diverse mechanisms also acting in either a positive or negative manner. This can be seen in the following

listing, in which several of the factors mentioned exert their influence, at least in part, on senescence.

A. HORMONE LEVELS

Rust researchers have long been intrigued by plant growth hormones because their effects on solute accumulation in cereals mimic, at least superficially, some of those exhibited by rust diseases. From these observations it has been postulated that the fungus in some way regulates hormone levels at and around the infection site so that solute accumulation is favored. Illustrative of this are the cytokinins, which by themselves can substantially alter translocation patterns by reducing the mobilization of reserves and their export from treated areas, especially in senescing leaves (Gilbert *et al.*, 1980). The result of this is a green zone surrounded by senescing, bleaching tissue. A phenomenon of similar appearance, called "green islands," is commonly observed in leaves infected by obligate parasites (Bushnell, 1967; Bushnell, Chapter 15, this volume).

Cytokinins apparently exert their delaying influence on senescence in part through a depression of specific enzyme systems involved in membrane function and solute transport (Gilbert *et al.*, 1980). Alterations in the balances among these enzymes, brought about either by changes in cytokinin levels or by some other factor that alters enzyme levels or their activities, might be involved in rust diseases. However, whereas accumulation in green islands on detached leaves can involve a net import, net accumulation in attached leaves is not very great, especially when compared with the amounts of nutrients going into spores at the infection site (Bushnell, 1967). Still unanswered is whether the changes resembling those induced by cytokinins have any role in making infected tissue such an effective sink.

Cytokinins as well as auxins and other growth hormones are known to undergo large increases in rusted tissues of cereals, although the reasons for the increases are not known (Bushnell, Chapter 15, this volume). Presumably these changes in the levels of plant growth hormones, particularly of cytokinins and auxins, may be involved with maintaining the host tissues adjacent to the pustule in a juvenile state such that senescence and its associated shift to an exporting status is both delayed and decreased in intensity. Whether the newly produced hormones' originate in the host or the parasite is uncertain. We know essentially nothing about how the pathogen might initiate host re-

sponses that could lead the host to produce elevated hormone levels, or whether the host can affect the pathogen's ability to produce these hormones and/or degrade them. Certainly, the complex changes in growth hormones deserve further study in relation to their role in controlling translocation processes in rust diseases.

B. TOXINS

Sempio (1959) has called attention to various ways in which toxins might be responsible for creating nutrient imbalances. They include (1) impaired transport, (2) changes in membrane permeability, (3) inhibition of metabolite synthesis, and (4) inhibition of metabolite utilization. If, in rust diseases, toxins produced by the pathogen are indeed important for creating such effects, they probably are acting quite close to and on either side of the host-parasite interface (see next section). There is no experimental evidence to show that toxins are translocated any distance away from the infection site, although by analogy with those from other pathogens, this is conceivable (Durbin, 1981).

C. MEMBRANE STRUCTURE

Currently, major advances are being made in our understanding of membrane structure as well as the driving forces responsible for the transmembrane movement of various types of solutes. From work done in this field, it seems possible that rust fungi may synthesize proteins or other compounds that can increase the membrane permeability of the host cell by inducing its plasmalemma to pass through a sequence of conformational states. These changes in state can be effected by molecules that bind either noncovalently (i.e., allosteric effectors) or by covalent bonding to functional groups present on the membrane surface. Evidence for this type of mechanism has been found with tobacco mosaic virus coat protein (Banerjee *et al.*, 1981), peptide hormones (Poss *et al.*, 1978), and toxins (Sessa *et al.*, 1969), all of which interact with and destabilize membranes. Such alterations are known to modulate the ionic conductance of membranes, as for example in vision (Montal *et al.*, 1977) and egg fertilization (Ridgeway *et al.*, 1977). Similar membrane effects could be produced by toxins or other compounds acting as ionophores (Durbin, 1981). The possibility that obligate parasites may alter membrane composition and/or structure is suggested by recent work on barley powdery mildew. Changes

in the pattern of cell plasmolysis and plasmalemma permeability after infection have led to the postulation that infection alters the neutral lipids but not the phospholipids of the plasmalemma (Lee-Stadelmann *et al.*, 1982).

The classical work of Thatcher (1939, 1942, 1943) showed that infection of susceptible, but not resistant, wheat cultivars by *Puccinia graminis* f. sp. *tritici* caused an increase in host cell permeability to several nonelectrolytes as well as to water. In this connection, we now know that various types of compounds enter plant cells via different portals. Some utilize the phospholipid bilayer, whereas others are transported by proteins. Consequently, it is an oversimplification to speak of a general increase in the permeability of host cells. Rather, the different pathways for transmembrane movement should be examined separately to see what role they might play in the effect Thatcher observed. Obviously, further study of this phenomenon could be very informative.

D. ENZYME ACTIVITIES

The pathogen may also be able to initiate other changes in the host membrane that could aid in its acquisition of nutrients. For example, Borochoy *et al.* (1982) have suggested that senescence may be controlled by membrane fluidity. They found that the fluidity of the lipid core decreased with age because of a reduced capacity of the plant cells to synthesize membrane phospholipids and their enhanced capacity to degrade them via phospholipase A. When this happens the membrane becomes "leaky." A similar phenomenon in rust infections could be a contributing factor to solute translocation out of plant cells (Gilbert *et al.*, 1980), or for the alterations described by Thatcher.

Enzymes involved in the transmembrane movement of solutes might be another example. For example, changes in membrane-bound transport ATPases appear to be important factors for solute transfer across the haustorial-plasmalemmal interface. According to Spencer-Phillips and Gay (1981) in their studies on bean rust, the host's plasma membrane in the structural domain of the haustoria [i.e., extra-haustorial region bounded by the haustorial neckband] lacks normal ATPase activity. There was also no evidence of ATPase activity in the haustorial plasma membrane. Thus transport across this portion of the host-parasite interface is thought to be passive.

Still another possibility involves changes in the activities of en-

zymes responsible for the synthesis and degradation of translocatable metabolites. Such changes, if they occur under the influence of the rust fungus, could play a significant role in determining source-sink activities. For instance, reactions that lead to the removal of sucrose at the infection site (i.e., the sink) would increase translocation, because the sucrose gradient between the site and its sources has now been increased. Thus increases in invertase and amylase activities noted in rusted tissues may be particularly pertinent. In the former case, Clancy and Coffey (1980) found up to a 24-fold increase for this enzyme in rusted flax leaves. Although the cellular location of these alterations is not known, their net result would be to enhance the accumulation of soluble substances in the infected regions. Also possibly related to this problem are the findings that *Uromyces phaseoli* produces an activator of β -amylase (Schipper and Mirocha, 1969), and *Puccinia recondita* causes both a localized and systemic activation of peptidases (Huber, 1978).

Some years ago, Atkin and Neilands (1972) showed that various siderophores would induce the formation of green islands. They postulated that in rust diseases these substances might play a role in the formation of this symptom by complexing iron and transporting it into the fungus at the host's expense. The result of this could be a massive alteration in the content of iron-containing enzymes in the host. Unfortunately, this idea has not been further studied.

E. WATER POTENTIAL

Nutrients are withdrawn from water-stressed tissues at a reduced rate (Hocking, 1982). Thus, if a rusted leaf is under water stress, the stress of itself will tend to reduce nutrient export from that leaf. This reduction could, in turn, slow protein degradation, and hence delay senescence of the leaf, if the two processes were linked by some kind of feedback mechanism. Because water potential is a central factor in controlling the plant's biochemical and physiological processes, many of the alterations observed with rust infection may basically result from changes in water potential (Duniway, 1976). A pressure gradient could be the driving force for movement toward the rust fungus. Likewise, osmotic uptake of water by the pathogen could be required for solute translocation within the fungus. Unfortunately, we do not know enough about the influence of rusts on the water relations of the host. Such information would be very pertinent for determining to what extent water potential may be linked to solute imbalances.

V. Applications

Tolerance can be defined as the ability of a plant to yield more than would normally be expected considering the amount of disease present. Under this general heading there appear to be grouped a number of diverse physiological phenomena that contribute to disease tolerance in different ways. Some of these ways involve interactions with the mechanisms governing translocation. In some cases tolerance appears to be due to the host's ability to continue to "fill" the developing grain in spite of a moderate to high number of pustules of an infection type that ordinarily would categorize the cultivar as susceptible. In other cases the pathogen's development is obviously retarded. Such cultivars are referred to as "slow-rusting" types. Here, the host appears to be able to divert nutrients away from the pathogen to the extent that the pathogen's rate of development is markedly reduced. Perhaps in these cultivars the developing grain is such an effective sink vis à vis the rust infection that normal translocation patterns are largely maintained. Alternatively, or additionally, the host may be producing some factor(s) that in some way hinders substrate utilization by the fungus or specifically delays its sporulation.

In some cultivars the flag leaf, glumes, and/or awns contribute substantial amounts of photosynthate to the developing grain (Durbin, 1967). Because of their proximity to the developing grain, they develop very strong source-sink relationships with the grain. Also, such structures, being younger than the remainder of the shoot, tend to be more lightly infected and hence less subject to the "sink effect" of rust infections. Even more emphasis needs to be placed on exploiting this important type of tolerance.

We need to identify the kinds of physiological mechanisms that operate in these cases and determine how they might be used to minimize disease losses. At present, it is not clear if restricting the pathogen's influence on translocation patterns can be a cause of host resistance, or whether it merely reflects an effect following from the expression of some other resistance mechanism. Still, it is feasible to look for mechanisms and substances that could act on sources and/or sinks, and upset the pathogen's effect on translocation patterns (i.e., reorder the priorities for solute distribution). Another strategy to minimize rust effects might be to develop cultivars with extended or shifted heading periods. If we could somehow manipulate these host processes, we might be on the threshold of developing a very useful control procedure.

VI. Conclusion

Although our understanding is incomplete on major aspects of how solute distribution patterns are altered by rust fungi, there is a growing body of information indicating that this phenomenon is central to controlling their growth potential, and that the effect of solute redistribution in the host constitutes one of the major ways in which rusts affect productivity, and conversely that solute redistribution can be a controlling factor in the growth of the pathogen. Assuming that these views are sustained, we should direct increasing efforts toward the study of solute redistribution mechanisms, employing a coordinated effort by diverse disciplines to develop control strategies.

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